ARTICLE IV.

OBSERVATIONS ON CHOKED DISK FOLLOWING INJURIES TO THE HEAD IN CHILDREN, WITH CASES. By CHARLES S. BULL, M.D., Surgeon to the N. Y. Eye Infirmary and to Charity Hospital.

INSTANCES of recovery from meningitis following injuries of the head are sufficiently uncommon to make four cases of this affection, occurring in children, prove of some interest. It is a surgical axiom that all injuries of the head may lead to meningitis sooner or later, and in cases of injury terminating fatally, as the large majority of such cases do, the autopsy frequently enables us to trace the course of the inflammation from the seat of injury, the scalp, through the dura mater to the arachnoid and pia mater, and thence to the brain substance. But this, I am inclined to think, is unusual, unless the bones of the skull are affected. If the disease commences in the dura mater, the inflammatory action is usually limited to that portion of the membrane in the neighbourhood of the injury, though inflammation of the dura with an effusion between it and the bone, without implication of its under surface, is very rare. If the whole thickness of the dura becomes involved, then the arachnoid and pia mater are attacked, and the inflammatory action extends rapidly in every direction. When an exudation takes place into the cavity of the arachnoid, it does not always extend, according to the statements of authorities upon the subject, beyond the upper and lateral surfaces of the brain; that is, it is not common to find any effusion at the base of the skull. Even when the pia mater is involved on the convexity, the exudation does not usually extend as far as the base of the brain, though we do sometimes meet with it here. This seems to be what is met with in autopsies of cases of meningeal inflammation arising from injuries to the head. According to Hutchinson, arachnitis with exudation into the cavity of the membrane is distinct from that variety of meningitis where the effusion takes place into the subarachnoid spaces. He thinks the two forms occur in connection with different causes and rarely complicate each other. According to him, inflammation of the pia generally begins at the base, and inflammation of the arachnoid on the convexity. The former usually results from laceration of a nerve, and the latter from lesion of the bone and dura mater. Hutchinson also affirms that in inflammation of the subarachnoid spaces we almost invariably have a fracture at the base of the skull, implicating one of the nerve canals; and that along the trunk of the latter, external to the true arachnoid cavity, the inflammation travels up to the subarachnoid spaces. Here inflammation often involves the neurilemma of nerves and gives rise to neuritis. In these cases the fixed dilated pupils may possibly be due to an inflammation about the roots of the third nerve as well as to optic neuritis. It is a well-known fact that fixed and dilated pupils are common in the more chronic cases of optic neuritis met with in connection with brain disease, though they are not always seen here.

According to other authorities, in the majority of cases of traumatic meningitis, the exudation, whether serous, fibrinous, or purulent, is found in the cavity of the arachnoid when the bone is injured; but that where the bone is not injured the effusion is usually upon the surface of the arachnoid. These statements would seem to recognize two kinds of traumatic meningitis; the one starting from a fractured or diseased bone and accompanied by exudation into the arachnoid cavity, the other starting from the brain as the result of concussion and accompanied by exudation upon and in the meshes of the pia mater. The view taken by Hutchinson seems the more reasonable of the two.

The question of diagnosis between the two forms of meningitis seems to me a matter of considerable doubt. Unless the presence of diseased bone could be demonstrated through the scalp wound it is scarcely probable that in a given case we could decide, from a study of the symptoms, whether the inflammatory action started from the bone and dura mater or from the brain and pia mater as the direct result of concussion. Almost all cases of serious lesions of the head manifest signs of concussion more or less plainly marked, though it is true that the reactionary inflammatory symptoms of concussion usually show themselves within a very short period after the injury.

When these cases terminate favourably, the exudation is probably changed into fibrous tissue, the pia mater becomes much denser in structure and adheres to the visceral layer of the arachnoid and to the brain substance. From some of the resulting symptoms or effects we conclude that considerable effusion of lymph has taken place at the base and across the longitudinal fissure, and is most abundant near the pons and about the optic nerves or chiasm. It is well known that a meningitis, either primarily or coincidentally with some other central lesion, is very frequently the cause of neuro-retinitis. Of course, basilar meningitis, with an exudation around or upon the optic nerves, chiasm, or tracts, is the most important intracranial cause; but it should be remembered that meningitis of the convexity of the hemispheres may also give rise to optic neuritis. *Berthold* thinks that according to the intensity of the extended inflammation and the consequent exudation into the lymph spaces of the eye, we have either a filling of Tenon's capsule and chemosis of the conjunctiva or a filling of the subvaginal nerve space, with choked disk or optic neuritis. Whatever may be said of the latter view it is about certain that conjunctival chemosis has no *direct* connection with intracranial inflammation of any form.

It has been asserted that inflammation of the brain substance and of the meninges present different symptoms when occurring at the base of the brain or on its convexity, and that it is possible to distinguish meningitis of the base from that of the convexity, chiefly from the influence which the diseased meninges exert on the functions of the parts beneath, such as the organs of special sense. But the irregularity in order of occurrence of the functional disorders, as well as their inconstancy, makes it very difficult at times to estimate the actual value of these symptoms, individually or collectively, as indicative of some localized brain trouble. We know that, leaving acute meningitis out of the question, chronic or subacute meningitis may occur a few days after the infliction of the injury, or not until months have elapsed, and the patient has apparently entirely recovered. Still even in cases where its appearance has been so long postponed, if we examine the patients carefully, some impairment of function, such as deafness, or amblyopia, or loss of some mental function, may generally be detected. An accurate, minute study of cases of injury to the head always yields some practical results. There are always at least three points to settle, as far as may be, in these cases: 1st. The situation of the lesion. 2d. What the lesion is. 3d. What effect has been produced upon the anatomical condition of the part itself and its immediate surroundings. It must be considered that in all cases of injury to the head there is more or less severe concussion of the brain substance, which is an important factor in the prognosis, for it is by no means uncommonly the real cause of death.

The subject of the ophthalmoscopic evidences of intracranial lesions and their value as aids in localizing the disease is still unsettled, and cannot be discussed satisfactorily within the limits of a short paper. It is doubtful whether any of the so-called ophthalmoscopic signs of intracranial inflammation are of much value as showing the *assured* presence of such inflammation, or the predominance of inflammatory action over obstructive changes. It is still uncertain whether we can always make a reliable differential diagnosis between the several conditions which may exist singly or in combination. Choked disk without impairment of vision may lead to atrophy of the optic nerves and total blindness. A choked disk may present very different appearances at different times, or at the same time in the two eyes, owing to conditions which exist entirely independent of brain disease. Moreover, it may not be the symptom of any intracranial disorder, but may be due to some affection of the optic nerve within the orbit, or even of the disk itself; and if we once admit this, of course the presence of choked disk ceases to be of any importance as an aid in localizing an intracranial lesion.

Case I.—Terence B., æt. 6; first seen October 12, 1874. Three months before he had received a severe blow on right temple, from which hemorrhage was profuse, but child did not lose consciousness, and was able to walk into house. One week later he fell from a wagon, and struck on same region of head. October 8th, mother first noticed that child could not see well. For four weeks after second accident child complained almost constantly of headache, and would frequently cry out at night. Since then has not complained much, but has appeared stupid and heavy. Vision has failed very rapidly during past week. Pupils moderately dilated and immovable. No strabismus. Typical example of choked-disk in both eyes; enormous swelling of optic papillæ, and distension of veins, but no retinal hemorrhages. Peculiar yellow, glistening exudation in region of macula in both eyes, and masses of exudation between macula and disk. No perception of light. Child complains of nothing except blindness, but passes large quantities of dark-coloured, offensive urine, which, on examination, is found filled with phosphates and amorphous urates. Heart and lungs normal. Child very quiet and stupid, with constant tendency to somnolence. Any treatment was probably useless, but an attempt was made to improve the vision by local bleedings, blisters to temple and behind the ear, and large doses of potass. iodid., but all proved of no avail.

In this case it is not possible to say whether the first or second injury gave rise to the brain trouble. The affection was without doubt a meningitis, probably at first of the convexity of the right cerebral hemisphere; but subsequently spreading, and possibly reaching the base, involving the optic chiasm, or the optic nerves in front of it. The exudation was probably not very extensive, since there was no paralysis of any other cranial nerves. The severe pain in the head, the screaming, and the later stupidity and tendency to sleep suggest meningitis as the most probable cause. Yet the little patient was never ill enough to be confined to his bed, and most of the time was moving about the room. The physician in attendance evidently suspected nothing of a serious nature, and the progress of the inflammation must have been insidious and of the subacute type, for the mother only noticed that the child could not see four days before I saw him. No ophthalmoscopic examination had been previously made. It is not probable that we can look to the concussion of the brain as the cause of the choked disk, unless indirectly through its setting up inflammation in the pia mater and arachnoid, which, in this case, is improbable. The choked disk was the result of compression by exudation, direct or indirect, and will end in atrophy of the optic nerve fibres. There is probably also some interstitial neuritis going on in all cases of choked disk, which may also aid in the atrophic process. The yellowish exudation in the macula lutea, though resembling that seen in cases of retinitis accompanying Bright's disease, is now regarded as by no means pathognomonic, as it is met with occasionally where no renal trouble exists. The child's health is perfectly restored. The obstructive and atrophic process in the optic nerves had evidently existed for some time before any failure of vision was noticed by the mother. The amblyopia may have begun very early after the accident, for slight degrees of choked disk or neuritis are often overlooked, owing to the disturbed sensorium of the patient. In view of the yellowish exudation in the region of the macula, suspicions were excited of a possible latent kidney affection; but though the urine was carefully examined at intervals of three or four days for a period of about four months, both chemically and microscopically, nothing abnormal was ever found. Moreover, the child is still under observation, and has never developed any other signs of renal disease.

CASE II.—John L., æt. 9; admitted to the New York Eye Infirmary August 28, 1876. Nine months before, the child had a heavy fall down stairs, struck on his head upon a stone pavement, but did not lose consciousness. There was a long, ragged cut over the occiput through the scalp, from which the hemorrhage was profuse. Four days later he began to complain of headache, which rapidly became very severe; vertigo ensued, followed by nausea and vomiting, and a high fever. The child did not, however, at any time, take to his bed, though at times he would scream loudly from the pain in his head. There were never any convulsions or signs of paralysis. The headache and other head symptoms gradually subsided, and the boy was apparently well until about three months after the accident, when he complained to his mother that the sight of the *left* eye was failing. No physician was called in, and the vision gradually faded away until the eye was perfectly blind. About two months before I saw him the vision in the *right* eye began to fail, and has steadily grown worse. The child is a bright, active, well-nourished lad. Both pupils are moderately dilated, but the left one is absolutely immovable. L. E., V. $=0$: total amaurosis. R. E., V. $=\frac{10}{200}$. The ophthalmoscope showed in the L. E. a brilliant white optic papilla, with very small arteries. In the R. E. there were all the signs of choked disk, with a most remarkable development of vessels upon the disk and retina, simulating in appearance an aneurism by anastomosis.

Here was probably a basilar meningitis set up by the injury, though of limited extent, as no cranial nerves were involved, save the optic. The process must have been subacute and slow, for it was not till three months after the accident that the child complained of failing vision in the *left* eye. The explanation of this is probably as follows: There was an exudation poured out over the course of the optic nerves or chiasm, which subsequently became organized and slowly underwent contraction. As contraction went on compression was exerted upon first the left optic nerve, interfering with the circulation and giving rise to choked disk, and the compression continuing, it resulted in the brilliant white atrophy which I saw at the boy's first visit. At a much later period vision began to fail in the right eye, showing that the exudation was still undergoing contraction and compression was being exerted upon the right optic nerve. Moreover this process was a very slow one, for two months after vision had begun to fail in this eye, there were all the signs of choked disk present.

Here was another case where the child was deemed so little ill that no physician was considered necessary, and it is probable that no interference could have prevented the subsequent atrophy of the optic nerves. As there was still some vision in the right eye, an attempt was made to preserve it, though with very little hope. Six leeches were applied to the temple, and this was repeated every fourth day for three weeks. Large doses of potass. iodid. were given, reaching as high as two drachms daily for several weeks. Blisters to the temple, and back of the neck were frequently used; but in spite of all, vision gradually faded out, and in less than a month after I saw him the atrophic process was well established in this nerve also.

Case III.—Mary E. S., æt. 8; admitted to the New York Eye Infirmary June 3, 1876. The child was perfectly well up to four years of age, when she received a violent blow on the back from a fall, since which time she has never walked. Just before Christmas, 1875, she fell down stairs some distance and struck on her head. She was picked up unconscious, but there was no external wound. She regained consciousness shortly, but two days later symptoms of meningitis showed themselves, marked by great pain in the head, vomiting, loud screaming, and frequent convulsions. The child was dangerously ill for three weeks, and has never fully recovered. She still complains of great pain over the occiput. Six weeks before I saw her the mother noticed that the child could not see, and now thinks that she is entirely blind. During the last two weeks deafness has appeared.

At present there seems to be an entire arrest of development of the whole body. The patient is no larger than a child of four years of age. There is complete paralysis of the lower extremities, caries of the lower dorsal and upper lumbar regions of the vertebræ, and general emaciation. Both pupils are widely dilated and immovable, the media clear, but both optic papillæ are very much swollen, a mass of exudation, and the disks undergoing atrophic degeneration. Total amaurosis, marked deafness, but no paresis of any of the ocular muscles.

Whether the meningeal inflammation in this case started from the convexity or the base cannot be determined, though from the delirium and convulsions forming so prominent features it may have arisen on the surface. Still, as the auditory nerves were evidently involved, some exudation at the base is highly probable independently of the condition of the optic disks.

Case IV.—Mary D., æt. 4; admitted to the New York Eye Infirmary September 14, 1876. About two months before admission the child, while playing upon the stairs, fell over the railing to the hall below, a distance of about eight feet, and struck upon her head. The child was stunned, but soon recovered and began to scream. There was a ragged, irregular scalp wound over the right frontal protuberance about an inch long, which bled profusely. The child gradually became quiet, and cold applications to the scalp controlled the hemorrhage. Next day she was playing about as usual, was apparently perfectly well, and the mother noticed nothing wrong until about five weeks after the accident. The child then began to grow stupid, showed a constant tendency to sleep, vomited her food, and staggered when she walked. She occasionally complained of headache, and had considerable fever, but was never sick enough to go to bed, and no physician was called in. The child was cross, but was never delirious or made any noise. About a week before I saw her the mother thought the child's eyesight was affected, because she ran against the furniture in the room.

On admission the child looked perfectly well in body, answered questions readily; was evidently a bright child, but with a peculiar stare in the eyes. The pupils were moderately and equally dilated and freely movable. Media perfectly clear. In right eye pronounced white atrophy of optic disk, arteries very much reduced in calibre, broad shallow excavation, and no signs of any preceding inflammation of nerve or retina. In left eye still some traces of choked disk, the papilla being still swollen below and the vessels engorged and tortuous, but the upper portion of the disk the seat of progressive atrophy. Absolute amaurosis of both eyes—not the faintest perception of light. The scar on the forehead was still visible, but was not sensitive on pressure, and will, probably, entirely disappear. The urine was examined, and gave sp. gr. 1020, acid reaction, and deposited abundant phosphates.

Large doses of potass. iodid. were given to this child for a period of three weeks, and a daily hypodermic injection of strychnia nitr. commencing with gr. $\frac{1}{120}$ and increasing the dose to gr. $\frac{1}{60}$, but with absolutely no effect upon the vision. On October 10, the patient was suddenly taken with high fever, violent headache, and screaming, but there was no vomiting or diarrhœa. The fever and headache lasted four days, and then the child as suddenly recovered.

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ARTICLE V.[1]

ON THE FUNCTIONS OF THE CEREBRAL LOBES. By B. F. LAUTENBACH, M.D., Demonstrator of Physiology in the University of Pennsylvania.

THE extirpation of the lobes of the cerebrum is a comparatively easy operation which in previous times has been made by Flourens,[2] Magendie,[3] Hertwig,[4] Longet,[5] Schiff,[6] and others. These older investigators arrived at the conclusion, that, following this operation, the movements are either not affected or only evanescently weakened. The functions depending on the volition and perception are completely abolished. Flourens found that pigeons with both hemispheres removed no longer fear fire. The irritation produced by pinching, pricking, or even burning the operated animal, causes it to be agitated slightly. If the irritant, however, be a second time brought towards the animal it fears it not, and makes no effort to escape from it. These animals have evidently lost their power to memorize the impressions received. The author has frequently observed that cats, in whom the cerebral lobes had been removed, no longer manifest the usual signs of fear and of extreme agitation when a dog approached them. Flourens concludes that memory, vision, audition, and volition, disappear with the removal of the hemispheres.

Schiff believed that the extirpation of the peripheral brain-substance only prevents the occurrence of those movements which proceed from excitation of the higher senses.

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[1] This paper was read before the Alumni Association of the Auxiliary Department of the University of Pennsylvania.

[2] Fonctions du Système Nerveux, Paris, 1842, pp. 31–37.

[3] Leçons s. l. Fonct. d. Syst. Nerv., Paris, 1839.

[4] Exper. de effect. lesion. in partibus Encephali, Berol., 1826.

[5] Traité de Physiologie, tome 2, part 2.

[6] Lehrb. d. Muskel u. Nervenphysiologie, Lahr. 1859.